Even though, the utility of the heparin in pts with unstable angina is well established, these results show the controversial role of postponing an angioplasty using the pre-treatment with IV heparin for 48 to 96 hrs. Other trials are necessary to evaluate the clinical and economic impact.

1190-103 The Contribution of Adjunctive Stent Use to In-patient Cost With and Without GPIIa/IIb Blockade in the RESTORE Trial

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RESTORE (Randomized Efficacy of Study of Tirofiban for Outcomes and Restenosis) was a randomized, double-blind, placebo controlled trial of tirofiban in 2,141 patients undergoing PTCA within 72 hours of presentation with unstable angina or acute myocardial infarction (MI). Patients were treated with tirofiban 10 ug/kg over 3 minutes followed by a 36 hour infusion of 0.15 ug/kg/min or placebo. At 2 days there was a 38% reduction (8.7% vs 5.4%, p = 0.005) in the composite endpoint of death, non-fatal MI, coronary surgery due to PTCA failure or recurrent ischemia, repeat target vessel PTCA for recurrent ischemia, or stent placement for actual or threatened abrupt closure of the dilated artery. A cost substudy was conducted in 818 patients at 30 sites. 3.9% of patients had stents placed as a "bail-out" for acute closure and 6.1% had stents placed due to an inadequate result. No patients had planned elective stenting in RESTORE. Hospital cost was $9,952 without stents, $14,160 with 1 or more stents. The additional cost of stents, accounting for other significant variables was $2,905 ± 737. The additional cost of other multivariate correlates were $19,103 for CABG, $5,446 for congestive failure, $7,106 for additional PTCA, $6,305 for a myocardial infarction, $5,334 for transfusion requirement and $1,047 for a first PTCA (R Sq 0.34). There was a trend to lower costs with tirofiban both in the stented and unstented patients. Patients receiving tirofiban had a mean hospital cost of $9,759 ± 5,508 without a stent and $13,862 ± 5,221 with a stent. Patients not receiving tirofiban had a mean hospital cost of $10,141 ± 6,366 without a stent and $14,339 ± 6,156 with a stent. Used for bailout after acute closure or in the setting of an inadequate result, even without anti-coagulation with coumadin, stents will incrementally add to the hospital cost of PTCA.

1190-104 Can Ticlopidine Be Safely Discontinued Two Weeks After Coronary Stent Placement?

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To determine the frequency of stent thrombosis (ST) and other adverse events 15 to 30 days after coronary stent placement when ticlopidine is stopped after 14 days, we analyzed 489 pts who underwent successful stent placement between 5/96 and 3/97. Pts on chronic warfarin, in shock, and in protocols requiring 4 weeks of tic-lopidine were excluded; ticlopidine was stopped after 2 weeks in all other pts (n = 489). Mean age was 64 yrs; 47% had suffered MI, 20% had undergone CABG, 63% had multivessel disease. Indications for the stents were dissection/abrupt closure (33%), suboptimal balloon angioplasty (16%), and elective (51%). The 489 pts received J & J coronary (76%), GR (21%), and J & J biliary stents (3%); 1.5 stents per pt were placed. Mean nominal stent size was 3.3 ± 0.6 mm. High-pressure inflations (≥12 atm) were performed in all pts (mean 17 ± 4 atm). Mean residual stenosis was 4 ± 6% (visual estimate). Pts at increased risk of stent thrombosis (14%) were also treated with Enoxaparin 30–60 mg SQ for 10–14 days.

Results: Adverse events ≤14 days after stent placement occurred in 9 pts (1.8%); 5 pts died and ST occurred in 4 others. However, there were no deaths, MI's, CABG or PTCA procedures, and ST did not occur, on the 15th–30th days. Neutropenia did not occur in any pt.

Conclusions: Stopping ticlopidine 14 days after stent placement is associated with a very low frequency of ST (0% in this study), less than the 1% frequency of life-threatening ticlopidine-induced neutropenia.

1190-105 Evidence of Pharmacologic Preconditioning During PTCA by Intravenous Pre-treatment of ATP-sensitive K+ Channel Opener NICORANDIL

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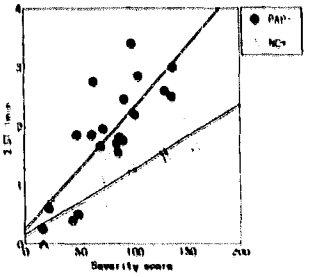
Background: To establish whether pretreatment of nicorandil (NC), ATP-sensitive K+ channel opener, induce preconditioning effect independent of the increase of collateral recruitment (F) in patients.

Methods: 28 pts with angina who underwent PTCA to proximal LAD stenosis were randomly allocated to pretreatment of NC (4 mg IV 5 min before initial ballooning) (n = 13) or conventional treatment (N = 14). Pts who experienced angina within 7 days before PTCA were excluded. ^{99m}Tc tetrofosmin were injected during balloon inflation, and quantitative analysis of ischemic severity during inflation was calculated (SS). ECG was recorded during 2 min inflation to calculate the sum of ST elevation (ΣST).

Results: Close correlation was observed between SS and Σ ST in both group (R2:control 0.77, NC 0.81) as shown in figure. NC resulted in significant suppression of ST elevation (SS adjusted Σ ST: Control 1.88 mV vs NC 1.09 mV $p < 0.01$). The line of regression in control is significantly steeper than NC, suggesting cell protective effect against ischemia by NC which is independent of flow variation.



Conclusion: NC pretreatment result in the induction of myocardial preconditioning independent of flow variation.

1190-106 Adenosine Preconditions Against Ischemia-Induced Systolic and Diastolic Dysfunction During Percutaneous Coronary Angioplasty

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Adenosine pretreatment increases the resistance of human myocardium to ischemia as assessed by ST-segment shifts, in a manner similar to ischemic preconditioning. It is unknown if either adenosine pretreatment or ischemic preconditioning protects the human heart against the adverse effects of ischemia on systolic or diastolic function. Thus, 18 pts having PTCA were randomized to receive a 10-min intracoronary infusion of saline (controls [C], n = 9) or adenosine-2 mg/min (ADO, n = 9). Ten min after infusion, pts had three 2-min inflations, each separated by 5 min. LV inflow propagation rate (LVPR) was measured at the end of each inflation by color M-mode echo. Wall motion was assessed by the centerline method (4- and 2-chamber).

Result: In C, LVPR decreased during each inflation, but progressively less from inflation 1 to 3. In contrast, in ADO the decreases in LVPR during inflation 1, 2 and 3 were similar. LVPR during inflation 1 was less in C vs ADO ($p < 0.05$) (table).

	LVPR (cm/s)				Chordal shortening (mm)			
Group	Base	Inf 1	Inf 2	Inf 3	Base	Inf 1	Inf 2	Inf 3
C	55 ± 4	27 ± 2[e]	30 ± 3[d]	36 ± 4[c]	8 ± 0.3	2.8 ± 0.3[f]	4.1 ± 0.4[f]	4.8 ± 0.5[f]
ADO	65 ± 8	40 ± 4[b]	38 ± 4[a]	37 ± 4[b]	8 ± 0.3	4.2 ± 0.4[f]	4.1 ± 0.4[f]	5.0 ± 0.3[f]

[a]p = 0.06, [b]p = 0.05, [c]p < 0.03, [d]p < 0.003, [e]p = 0.0003 vs Base & [f]p < 0.0003 vs Base. X ± SEM

In C, chordal shortening (CS) in the ischemic zone decreased during each inflation, but less so during inflations 2 and 3 compared to inflation 1 (both p = 0.0005). In ADO, the decreases in CS were similar for inflations 1, 2 and 3. CS during inflation 1 was greater in ADO vs C ($p < 0.02$). CS during inflation 3 in C was comparable to inflation 1 in ADO.

Conclusions: To our knowledge, this is the first study showing that adenosine preconditions human myocardium against ischemia-induced systolic and diastolic dysfunction. This is also the first evidence that ischemic preconditioning attenuates mechanical dysfunction during acute myocardial ischemia in man.